U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 20 September 2006

In the Matter of:

M.M., Widow of and on behalf of
A.M., deceased Miner,
Claimant

v.

BISHOP COAL COMPANY, INC.,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest

Case Nos.: 2004-BLA-06248
2004-BLA-00111

APPEARANCES:

Joseph E. Wolfe, Esquire
For the Claimant

Ashley M. Harman, Esquire
For the Employer

Before: EDWARD TERHUNE MILLER
Administrative Law Judge

DECISION AND ORDER – DENYING BENEFITS

Statement of the Case

This proceeding arises from claims for benefits filed by A. M., the now deceased Miner, and M.M., his surviving spouse, under the Black Lung Benefits Act, 30 U.S.C. §901, *et seq.* Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.[1] This claim is governed by the law of the United States Court

[1] All cited regulations refer to Title 20, Code of Federal Regulations, unless otherwise indicated, and are cited by part of section only. The Secretary of Labor adopted amendments to the "Regulations Implementing the Federal Coal Mine Health and Safety Act of 1969" as set forth in Federal Register/Vol. 65, No. 245 (Wed. Dec. 20, 2002). The revised Part 718 regulations became effective on January 19, 2001 and were to apply to both pending and newly

of Appeals for the Fourth Circuit, since the Claimant's last coal mine employment occurred in Virginia. *See Shupe v. Director, OWCP*, 12 B.L.R. 1-202 (1989)(en banc).

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

The Miner's (*duplicate*) claim was filed on March 28, 2000 (MDX 3). [2] The evidentiary limitations contained in the revised regulations do not apply in the Miner's claim (MDX 24). The pre-amendment provisions of §725.309 are applicable. Because the Widow's claim was filed on September 10, 2001, the evidentiary limitations set forth in §725.414 are applicable to that claim (WDX 2).

A formal hearing was held on July 19, 2005 in Pipestem, West Virginia. All parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations issued. The record was held open to allow for the submission of additional evidence and closing written arguments (TR 27-28, 54-55). Procedural orders dated August 29 and October 17, 2005, extended the deadlines for the submission of evidence and closing arguments. The record was closed on or about November 2, 2005, after receipt of the written closing arguments by the respective parties.

Since the evidentiary limitations do not apply to the Miner's claim, the record in the Miner's claim consists of the hearing transcript, Director's Exhibits 1 through 24 in the Miner's claim file (MDX 1-24), Director's Exhibits 1 through 40 in the Widow's claim file (WDX 1-40), Employer's Exhibits A (EX-A) and 1 through 15 for Miner's claim (MEX 1-15), Employer's Exhibits 1 through 13 for Widow's claim (WEX 1-13),[3] Claimant's Exhibits A (CX-A) and 1 through 5 (CX 1-5),[4] and Joint Exhibits 1 and 1A (JX 1 & 1A).[5]

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filed cases. The new Part 725 regulations also became effective on January 19, 2001. Some of the new procedural aspects of the Part 725 regulations only apply to claims filed on or after January 19, 2001 *not* to pending cases. For example, the evidentiary limitations set forth in §725.414 do not apply to the adjudication of claims pending on January 19, 2001. The "duplicate" or "additional" claims provisions in §725.309 are also not applicable to such pending claims.

[2] The District Director's office forwarded the Miner's claim file consisting of 24 Director's exhibits, and the Widow's claim file consisting of 40 Director's exhibits. The Director's exhibits contained in the Miner's claim file are identified herein as "MDX." The Director's exhibits contains in the Widow's claim file are identified herein as "WDX." Employer's exhibits for the Miner's claim have been marked as "MEX," and Employer's exhibits for the Widow's claim have been marked as "WEX."

[3] Employer's post-hearing submissions included supplemental reports by Drs. Rosenberg and Fino which have been marked as MEX 13 & 14 in the Miner's claim, and WEX 11 & 12 in the Widow's claim. Under cover letter dated September 6, 2005, Employer submitted the deposition transcript of Dr. Perper which has been marked as MEX 15 and WEX 13, in the Miner's and Widow's claims, respectively.

[4] Except for Claimant's Evidence Summary Form, which was marked and received as Claimant's Exhibit A (CX-A), and Dr. Perper's report, dated June 18, 2005, and curriculum vitae, which were marked and received as Claimant's Exhibit 3 (CX 3), separate exhibits were not submitted for Claimant's Exhibits 1, 2, 4, or 5. However, as discussed at the formal hearing and reiterated in Employer's closing argument, pp. 3-4, these Claimant's exhibits are contained in the Director's Exhibits.

Since the Widow's claim is subject to the evidentiary limitations set forth in the new regulations, the record in the Survivor's claim consists of the hearing transcript, Director's Exhibits 1 through 40 in the Widow's claim file (WDX 1-40), Employer's Exhibits A (EX-A) and 1 through 13 for Widow's claim (WEX 1-13), Claimant's Exhibits A (CX-A) and 1 through 5 (CX 1-5), and Joint Exhibits 1 and 1A (JX 1 & 1A). Director's Exhibits 1 through 24 in the Miner's claim file (MDX 1-24) and Employer's Exhibits 1 through 15 in the Miner's claim (MEX 1-15) are excluded.

Employer's Exhibits 1-10 in the Widow's claim (WEX 1-10) have been admitted of record, and as set forth in the joint stipulation of the parties (JX 1 & 1A). The post-hearing supplemental reports by Drs. Rosenberg and Fino dated August 8, and August 16, 2005, respectively, have been marked as WEX 11 and 12, and received in evidence as rehabilitative reports, since they address those parts of Dr. Perper's report dated June 18, 2005, which criticize their opinions (CX 3). Dr. Perper's deposition transcript dated August 15, 2005, has been marked and received as WEX 13.

Several of the physicians' reports and deposition testimony in evidence refer to evidence of the Claimant and Employer excluded from the Widow's case regarding consideration of such reports or testimony. The Benefits Review Board has stated that the options include redacting the objectionable content, excluding the reports, asking the doctors to submit new reports and factoring the fact of such revised submissions in when deciding the weight to be given to such reports. Exclusion is not a favored option. *See Harris v. Old Ben Coal Co.*, 23 BLR 1-_____ , BRB No. 04-0812 BLA (Jan. 27, 2006); *see also, Webber v. Peabody Coal Co.*, 23 BLR 1-_____, 05-0335 BLA (Jan. 27, 2006)(en banc). The regulations provide for an exception, which allows the inclusion of medical evidence in excess of the limitations contained in §727.414 upon a showing of good cause. *See* 20 C.F.R. §725.456(b)(1) and §725.414(d).

Under the particular facts of this case, in which the physicians' reports and/or deposition testimony are admissible in the Miner's pre-amendment claim, this tribunal would deem it be professionally irresponsible and intellectually dishonest for physicians to redact or exclude technically objectionable parts of their reports or testimony in respect of the Survivor's *post*-amendment claim. Accordingly, good cause is deemed to have been established for the inclusion of these exhibits in their entirety. Since the objectionable evidence is primarily older medical data which was obtained long before the Miner's death and in view of the progressive and irreversible nature of pneumoconiosis, such evidence tends to be less probative than the more recent medical evidence, as in this case. The older medical data, which was filed in conjunction with the living Miner's claim, does not directly address the death causation issue. Accordingly, the analytical focus of the Widow's case is the treatment records shortly before the Miner's death, and the medical opinion evidence which directly addresses the issue of death due to pneumoconiosis. Even if the objectionable material were excluded from the reports and physicians' deposition testimony, the exclusion of such objectionable material would not be outcome determinative.

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[5] The parties jointly submitted what appear to be duplicative summaries of the issues and evidentiary rulings, in letters dated August 2, 2005 and October 13, 2005, respectively. These have been marked and received as JX 1 and JX 1-A.

The findings of fact and conclusions of law which follow are based upon analysis of the record, including the documentary evidence admitted, testimony presented, and arguments of the parties. Where pertinent, credibility determinations have been made.

Procedural History

Miner's Claim

The Miner filed his first application for black lung benefits under the Act on September 11, 1986 (MDX 1). Following a formal hearing on January 11, 1989, Administrative Law Judge Ben L. O'Brien denied benefits on December 26, 1989. Judge O'Brien considered interpretations of various chest x-rays dated October 28, 1986 through December 20, 1988, which he found did not establish pneumoconiosis. He considered the medical opinions of Drs. Rasmussen, Carpenter, Crisalli, Qazi, Zaldivar, Fino, and Kress. Drs. Rasmussen, Carpenter, and Qazi had found a respiratory insufficiency related to coal dust exposure. The other physicians related the Miner's problems to asthma and/or cigarette smoking. Dr. Crisalli reported a diagnosis of pneumoconiosis, despite Judge O'Brien's statement to the contrary. In addition, Judge O'Brien considered pulmonary function studies and arterial blood gas tests during the period from November 17, 1977 to November 23, 1988. Almost all of the pulmonary function studies were qualifying; none of the arterial blood gas tests were qualifying. Based upon that evidence, Judge O'Brien found that the Miner "does not have pneumoconiosis and is not disabled by the disease." (MDX 1).

On appeal, the Benefits Review Board affirmed on July 29, 1991. The Board noted that Judge O'Brien had inaccurately stated that Dr. Crisalli's opinion does not support a finding of pneumoconiosis, but that this constituted harmless error because the Board affirmed Judge O'Brien's finding that the Miner is not totally disabled due to pneumoconiosis, noting that, despite Dr. Crisalli's diagnosis of pneumoconiosis, he attributed the Miner's significant respiratory impairment to asthma, not pneumoconiosis or coal dust exposure (MDX 1). The U.S. Court of Appeals for the Fourth Circuit affirmed *per curiam* on August 14, 1992, stating:

> The reasoned opinions of Drs. Crisalli and Zaldivar, based on improved post-dilator spirometer studies and other data, are probative evidence which contradicts the positive pulmonary function studies, and the opinions of Drs. Rasmussen, Qazi and Carpenter. The ALJ was within his discretion in according greater probative weight to Dr. Crisalli and Dr. Zaldivar's opinions in view of their well grounded analysis of the cause of [the miner's] disability….In affirming the ALJ, the Board reached rational conclusions based on substantial evidence.

(MDX 1, U.S. Court of Appeals Decision). Since the Miner took no further action the denial became final and the claim was administratively closed (MDX 1).

On April 12, 1995, the Miner filed another application for black lung benefits which was denied by the District Director on October 9, 1995 because the evidence did not establish pneumoconiosis and that the disease was caused at least in part by coal mine work or a material

change in conditions since the previous denial (MDX 2). By implication, the District Director found that the Miner had not established disability caused by pneumoconiosis, because to establish a material change in conditions in the Miner's current claim, the Miner must establish at least one of the following conditions of entitlement: the presence of pneumoconiosis; its causal relationship to coal mine employment; or that the Miner's total disability is due to pneumoconiosis. The District Director apparently found that the Miner had established the presence of a total pulmonary or respiratory disability.[6] Therefore, "total disability" cannot be the basis for finding a material change in conditions under §725.309.

The Miner took no further action within a year, the denial became final, and the claim was administratively closed (MDX 2).

On April 6, 2000, the Miner filed the current application for black lung benefits (MDX 3). On March 28, 2001, the District Director issued a Proposed Decision and Order denying benefits (MDX 17). By letter, dated April 23, 2001, the Miner filed a timely request for a formal hearing (MDX 18). In correspondence, dated August 7, 2001, Claimant, the Miner's Widow, advised the District Director's office that her husband had died, and indicated that she would pursue his claim as a substitute party (MDX 21). On or about May 11, 2004, the Miner's claim file was forwarded to the Office of Administrative Law Judges (MDX 22-24).

Widow's Claim

On September 10, 2001, Claimant filed an application for benefits as the Miner's surviving spouse (WDX 2). On March 5, 2004, the District Director issued a Proposed Decision and Order awarding benefits (WDX 33). Following Employer's timely requests for a formal hearing (DX 34, 35), the Widow's claim was forwarded to the Office of Administrative Law Judges (WDX 38-40).

Issues

Miner's Claim

I. Whether the Miner had pneumoconiosis as defined by the Act and the regulations?
II. Whether the Miner's pneumoconiosis arose out of coal mine employment?
III. Whether the Miner was totally disabled?
IV. Whether the Miner's disability was due to pneumoconiosis?
V. Whether the evidence establishes a material change in conditions under §725.309?

[6] Attached to the District Director's denial letter dated October 9, 1995, are the results of a pulmonary function study dated July 13, 1995, and arterial blood gases dated May 17, 1995. Although the exercise blood gas results are slightly above the qualifying values, the resting blood gas results are qualifying. The pulmonary function study results are also qualifying. Accordingly, the District Director stated: "NOTE: Although the results of the breathing test and/or the blood gas test meet the disability standards described above, the evidence does NOT establish that your impairment was caused by black lung disease." (MDX 2).

Widow's Claim

I. Whether the Miner had pneumoconiosis as defined by the Act and the regulations?
II. Whether the Miner's pneumoconiosis arose out of coal mine employment?
III. Whether the Miner's death was due to pneumoconiosis?

(JX 1 & 1A; TR 13-16).[7]

Findings of Fact

Background

A. Coal Miner and Length of Coal Mine Employment

The parties stipulated, and this tribunal finds, that the Miner engaged in coal mine employment for 35 years (TR 15). The Miner retired in October 1982, when the mines closed down (MDX 1).

B. Dependents

Employer does not dispute, and this tribunal finds, that the Miner's spouse is his sole dependent for the purpose of augmentation for benefits (TR 15). The Widow has no dependents (WDX 2).

C. Responsible Operator

Employer concedes, and this tribunal finds, that it is the properly designated responsible operator (TR 15).

D. Personal Background and History

The Miner was born on August 18, 1926 (MDX 3). He married Claimant on March 25, 1947 (MDX 6). They remained married until the Miner's death on July 24, 2001 (WDX 2, 8).

I. Miner's Claim

New Medical Evidence

The new medical evidence in the current duplicate Miner's claim includes various chest x-rays, pulmonary function studies, arterial blood gases, and physicians' opinions, which were obtained and submitted after the most recent denial of the Miner's claim on October 9, 1995 (MDX 2). Since total disability was *not* the basis for the final denial, in order for Claimant to establish a material change in conditions under §725.309, she must establish at least one of the

[7] The constitutionality and application of the Act and regulations are preserved by Employer for appellate purposes only (TR 14-15).

other elements of entitlement consisting of pneumoconiosis, causal relationship, and disability caused by pneumoconiosis previously adjudicated against the Miner. The new medical evidence is summarized below.

A. Chest X-rays

The case file includes a descriptive interpretation by Dr. Edward Schmidt, in which he found "COPD" on a chest x-ray dated September 22, 1999, noting that the findings are "unchanged from July 1997." (WDX 15). The finding of COPD neither precludes nor establishes the presence of pneumoconiosis, and Dr. Schmidt's x-ray reading does not conform to the classification requirements set forth in §718.102(b). Therefore, it is accorded little weight.

The record also includes multiple interpretations of a chest x-ray dated July 10, 2000, by Drs. Patel (WDX 12; CX 1), Alexander (MDX 15; WDX 12; CX 4), Zaldivar (WDX 9; MEX 1), Navani (MDX 16), Meyer (WDX 11; MEX 2; WEX 1), Spitz (WDX 10; MEX 2; WEX 1), and Wiot (WDX 10; MEX 2; WEX 1), respectively. Of the numerous substantive interpretations of that x-ray, only Dr. Patel's 1/0, s/s, all six lung zones reading, and, Dr. Alexander's 1/1, p/q, all six lung zones reading, are positive for pneumoconiosis. The other interpretations by Drs. Zaldivar, Navani, Meyer, Spitz, and Wiot, B-readers, and, except for Dr. Zaldivar, Board-certified radiologists, are negative for pneumoconiosis under §718.102(b). Since the clear majority of the recent x-ray interpretations in the Miner's case are negative for pneumoconiosis, pneumoconiosis has not been established based on the x-ray evidence.

B. Pulmonary Function Studies

The record contains numerous pulmonary function studies dated August 31, 1999 (WDX 15), September 22, 1999 (WDX 15), July 10, 2000 (WDX 12), and September 13, 2000 (WDX 15). For a person of the Miner's height (72") and age (73 or 74 years old) at time of testing, the qualifying values of such studies must be FEV1 of 2.04 *and* equal to or less than an FVC of 2.63, an MVV of 82, *or* an FEV1/FVC ratio equal to or less than 55%.

The results of the pulmonary function studies are conflicting. Although most of the pre-bronchodilator studies are qualifying, almost all of the post-bronchodilator studies are not qualifying. The results of the pulmonary function tests conducted on August 31, 1999, September 22, 1999, and July 10, 2000 reveal a significant improvement in the post-bronchodilator FEV1 result, but the September 13, 2000 pulmonary function test shows little change with the pre-bronchodilator FEV1 of 2.07 slightly above the qualifying standard, while the post-bronchodilator FEV1 of 2.04 meets the regulatory criteria.

Viewed as a whole, the pulmonary function study evidence neither precludes nor establishes the presence of a totally disabling pulmonary or respiratory impairment. Even assuming that "total disability" were found, it would not establish a material change in conditions under §725.309, since the absence of total disability was not the basis for the final denial of the prior claim. Moreover, most of the more recent pulmonary function studies of

record reveal an improvement in the test results which is inconsistent with the progressive and incurable nature of pneumoconiosis.

C. Arterial Blood Gas Studies

The record contains arterial blood gas studies dated September 22, 1999 (WDX 15) and July 10, 2000 (WDX 12). The former was conducted only at rest, while the latter study was administered at rest and with exercise. None of the recent arterial blood gas tests are qualifying under Part 718, Appendix C, and do not support a finding of total disability. The recent arterial blood gas studies yielded better resting and exercise results than those obtained on May 17, 1995, which is inconsistent with the progressive and incurable nature of pneumoconiosis (WDX 14).

D. Physicians' Opinions

The record includes medical records from Clinch Valley Physicians, Inc., consisting of numerous progress notes primarily by Dr. Larry G. Mitchell dated September 30, 1994 through June 18, 2001. In addition, there are several office notes by Dr. German Iosif, a pulmonary specialist, dated September 22, 1999 through September 21, 2000 (WDX 13, 14, 15, 18; CX 2). Many of the progress notes list coal worker's pneumoconiosis among various diagnosed conditions, which also include acute upper respiratory infection, chronic obstructive pulmonary disease, hypertension, impaired glucose tolerance, hypothyroidism, hyperlipidemia, and gout. However, the underlying rationale for the diagnosis of coal worker's pneumoconiosis is not well-reasoned or well-documented. Mostly the progress notes reveal "clear" lungs on examination and several of the office notes indicate considerable improvement following pulmonary rehabilitation, despite listing pneumoconiosis among the diagnosed conditions. On September 22, 1999, Dr. Iosif reported "a dramatic improvement of his [the Miner's] exertional dyspnea with a significant recovery of his endurance capabilities. He [the Miner] is happy to report that he has not felt as well from a respiratory standpoint in at least five years. There are no complaints of current cough or purulent sputum production." (WDX 15). The January 26, 2000 office note states, in pertinent part: "[The Miner] has completed his course of outpatient pulmonary rehab and reports significant improvement in his endurance and exertional capability." (WDX 15). On June 18, 2001, Dr. Mitchell stated, in pertinent part, that "with the use of his aerosol treatments which include albuterol and Atrovent, he is able to go about his normal household activities and enjoys activities such as working in his garden." (WDX 14). Such findings of dramatic or significant improvement are inconsistent with the progressive and incurable nature of pneumoconiosis, and so the diagnosis of coal worker's pneumoconiosis in the progress notes is accorded little weight.

With respect to the Miner's claim, the more relevant evidence includes Dr. Rasmussen's report dated July 10, 2000 (WDX 12), the hospital records at or about the time of the Miner's death (WDX 13; WEX 2), the Miner's death certificate (WDX 8), and, the medical opinions of Drs. Mitchell (WDX 18), Perper (CX 3; MEX 15), Zaldivar (WDX 17), Branscomb (MEX 4, 5), Rosenberg (MEX 6, 8, 12, 13), Castle (MEX 7, 8), and Fino (MEX 10, 11, 14).

Dr. D. L. Rasmussen, who is board-certified in Internal Medicine and Forensic Medicine, examined the Miner on July 10, 2000. He reported the Miner's coal mine employment history from 1947 to 1982, and the Miner's family, medical, and social histories. The latter included a smoking history of 1 pack per day beginning in 1943 and ending in 1998. Dr. Rasmussen also reported abnormal findings on physical examination of the lungs, as well as the following clinical tests results administered on July 10, 2000:

Chest X-ray	Pneumoconiosis s/s 1/0
Vent Study (PFS)	Moderate, partially reversible obstructive ventilatory impairment
Arterial Blood Gas	Normal

(WDX 12).

Dr. Rasmussen diagnosed coal workers pneumoconiosis (CWP) attributable to 35 years of coal mine employment and x-ray changes of pneumoconiosis; chronic obstructive pulmonary disease (COPD), indicated by chronic productive cough, airflow obstruction; probable and arteriosclerotic heart disease (ASHD) indicated by abnormal exercise induced chest pain. (WDX 12, Sec. D6). Dr. Rasmussen identified the etiology of the cardiopulmonary diagnoses and provide his rationale as "1. CWP - coal mine dust exposure 2. COPD – coal mine dust exposure and cigarette smoking. 3. ASHD – non occupational" (WDX 12, Sec. D7). He assessed the severity of Claimant's impairment from a chronic respiratory or pulmonary disease, if any, and the extent to which such impairment would prevent Claimant from performing his last coal mine job, opining, "The patient has minimal to moderate loss of lung functions. He does not retain the pulmonary capacity to perform his last coal mine job." (WDX 12, Sec. D8a). In a report dated July 29, 2000, Dr. Rasmussen reiterated his opinion,

> Overall, these studies indicate minimal to moderate loss of pulmonary function. The patient does not retain the pulmonary capacity to perform his last regular coal mine job.
>
> There are two primary risk factors for this patient's impaired function. These include his cigarette smoking and his coal mine dust exposure. The latter is a contributing factor.
>
> The patient also has an abnormal electrocardiogram. The patient was informed of the abnormality as was his personal physician. His physician was sent copies of his electrocardiographic records.

(WDX 12).

Significantly, Dr. Rasmussen's diagnosis of pneumoconiosis is based in large part upon a questionable positive chest x-ray reading. The text of his narrative report establishes that, not only were the blood gases normal, but the pulmonary function results were, at least, partially reversible after bronchodilator therapy. It is unclear in the absence of a credible positive x-ray reading for pneumoconiosis, whether Dr. Rasmussen would still have diagnosed pneumoconiosis or found that the Miner's COPD is related, at least partly, to coal mine dust exposure. Moreover, Dr. Rasmussen failed to explain adequately his rationale for relating the Miner's COPD to coal mine dust exposure. Dr. Rasmussen reached similar conclusions following his examination of

the Miner on April 17, 1987 (MDX 1, sub-exhibits 22, 24). Although he credited the total disability finding, Judge O'Brien rejected Dr. Rasmussen's diagnosis of pneumoconiosis and his conclusion that the impairment was related to coal mine dust exposure in favor of better reasoned and documented medical opinions by other physicians and was affirmed by the Benefits Review Board and the U.S. Court of Appeals for the Fourth Circuit (*See* MDX 1 – ALJ). Consequently, Dr. Rasmussen's opinion had been accorded little weight.

The Tazewell Community Hospital records establish that the Miner was seen in the emergency room on July 24, 2001, under a "Code Blue," in which he was in full cardiac arrest and unresponsive. Under past history, it was noted that the Miner had COPD and cardiac disease. Under "Clinical Impression," it was noted: that cardiopulmonary resuscitation had been unsuccessful; the Miner expired and was pronounced dead; and, "Possible MI, acute, massive." In addition, the "Record of Death" lists a diagnosis of "cardiopulmonary arrest" (WDX 13; WEX 2).

The Miner's death certificate, signed by Dr. Larry G. Mitchell, states that the Miner died on July 24, 2001, at age 74 (WDX 8). The immediate cause of death was reported as "Probable ventricular arrhythmia" due to "COPD (with) coalworker's pneumoconiosis." The interval between the Miner's probable ventricular arrhythmia and death was "immediate." Dr. Mitchell recorded that the "COPD (with) coalworker's pneumoconiosis" occurred "years" before the Miner's death and listed other significant conditions contributing to death, but not resulting in the underlying cause as hypertension, hypothyroidism, and hyperlipidemia. No autopsy was conducted (WDX 8, Sec. 28a). Dr. Mitchell had treated the Miner for various conditions for several years prior to his death, but the death certificate, in and of itself, does not constitute a reasoned or documented medical opinion.

Dr. Larry G. Mitchell issued a one-page, "To Whom It May Concern" letter, dated August 17, 2002, which stated that he had been the Miner's primary care physician for "a number of years," having followed the patient from September 1994 until the Miner's death. The letter referenced the Miner's 35-year coal mine employment history, but did not mention his cigarette smoking history (WDX 18). Dr. Mitchell stated that he had treated the Miner with aerosol treatments of Albuterol, Atrovent, and Serevent, as well as oral medications including Humibid and Theophylline and noted that the Miner was on numerous other medications for other, unspecified medical problems. Dr. Mitchell opined:

> [The Miner] died from probably ventricular arrhythmias. He evidently died in his sleep. It is my opinion that his death was hastened by coal workers' pneumoconiosis since the pulmonary impairment associated with coal workers' pneumoconiosis and lack of oxygen definitely predisposes a person to ventricular arrhythmias. It is likely that he would have lived longer had his lungs not been compromised by the Black Lung.
>
> The effect of exposure to coal dust lead to the coal workers' pneumoconiosis which impacted his health negatively as described above.

(WDX 18).

Dr. Joshua A. Perper, who is Board-certified in Anatomical and Surgical Pathology, as well as Forensic Pathology, issued a lengthy report, dated June 18, 2005, in which he summarized the then available evidence (CX 3, pp. 1-24). Dr. Perper contended that the opinions of Employer's numerous consulting physicians regarding the absence of clinical or legal pneumoconiosis, disability causation due to pneumoconiosis, and/or death due to pneumoconiosis, were all flawed (CX 3, pp. 24-27). He addressed various "medicolegal questions" (CX 3, pp. 27-32). and attached an Appendix entitled "Coal workers' pneumoconiosis and associated centri-lobular emphysema" to his report (CX 3, pp. 3, pp. 33-39).

Dr. Perper found "significant and substantial coal worker's pneumoconiosis" based upon the following: a 35-year coal mine employment history, subjective and objective manifestations of respiratory disease, a number of radiological findings showing pneumoconiosis or granuloma, the progression and worsening of the Miner's respiratory disease despite the absence of cardiac disease, and clinical diagnoses of COPD, chronic bronchitis and coal worker's pneumoconiosis by Drs. Mitchell and Iosif (CX 3, pp. 27-28). Dr. Perper opined that coal worker's pneumoconiosis substantially contributed and/or accelerated the Miner's death based upon the following: a 30-plus year history of occupational exposure; clinical documentation of progressive respiratory symptoms; arteriosclerotic heart disease, mostly asymptomatic with no evidence of congestive heart failure; and, generally good control of hypertension (CX 3, pp. 31-32).

In summary, Dr. Perper set forth the following conclusions:

1. [The Miner] had evidence of significant and substantial coal workers' clinical and radiological pneumoconiosis.

2. [The Miner's] coal workers' pneumoconiosis was causally associated, at least in part, with significant COPD and emphysema.

3. [The Miner's] coal workers' pneumoconiosis was a result of longstanding occupational exposure of more than 35 years as a coal miner to mixed coal dust containing silica.

4. [The Miner's] coal workers' pneumoconiosis was a substantial contributing cause of his death and a hastening factor in his death.

(CX 3, p. 32). Dr. Perper reiterated the foregoing opinion in his testimony at his deposition on August 15, 2005 (WEX 13; MEX 15).

Dr. George L. Zaldivar, a B-reader who is board-certified in Pulmonary Diseases, Internal Medicine, Sleep Disorder, and Critical Care Medicine, issued a supplemental report, dated June 26, 2002, in which he reviewed the then available medical records (WDX 17). Dr. Zaldivar had previously examined the Miner on November 23, 1988 (WDX 16). Dr. Zaldivar set forth the Miner's coal mine employment history, his own findings as reported on December 15, 1988, Dr. Rasmussen's opinion and clinical test results on July 10, 2000, various chest x-ray

interpretations, the emergency room hospital records at the time of the Miner's death, and the death certificate. Dr. Zaldivar opined:

1. There is no evidence in this case to justify a diagnosis of coal worker's pneumoconiosis.

2. There was a respiratory impairment present. The impairment was a result of asthma, not only unrelated to his occupation as a coal miner, but, also, unrelated to his smoking habit which was, in itself, significant enough to potentially produce emphysema.

3. From the pulmonary standpoint, [the Miner] had a variable impairment which had been untreated for many years. The impairment as of the times he was examined in 1988 and again in the year 2000 was sufficient to prevent him from performing heavy manual labor which he said he was required to do. He could perform other labor on a continuous basis. By the year 2000, he also had coronary artery disease that would have prevented him from performing any work other than the sedentary level. Coronary artery disease is not related to his occupation.

4. Cold (sic) dust exposure did not play any role in his death. [The Miner] died as a result of coronary artery disease with either an acute myocardial infarction or a lethal arrhythmia causing the cardiac arrest.

5. Coal dust exposure did not play any role in [the Miner's] death. It did not cause nor contribute to his death.

6. Even if [the Miner] were found to have coal worker's pneumoconiosis by tissue sampling of his lungs, my opinion would remain exactly as given in this report.

(WDX 17).

In a supplemental report, dated August 23, 2004, Dr. Zaldivar reviewed various additional medical records, including the Miner's treatment records at Clinch Valley, the death certificate and medical opinions of Drs. Mitchell and Branscomb (WEX 7), and concluded, after considering this additional information:

1. There is no evidence in this case to justify a diagnosis of coal worker's pneumoconiosis.

2. There was a pulmonary impairment prior to death and this impairment was variable and it was the result of asthma, which is a disease of the population at large and unrelated to coal mining.

3. From the pulmonary standpoint, his ability to work was variable depending on the state of the asthma at any given time. Depending on the treatment, which he was receiving, as well as environmental factors, such as weather, cold air, etc., which cannot be controlled. The state of his lungs varied. At the time that I tested him he was capable of performing his usual coal mining work or work requiring similar exertion. (sic)

4. As a whole man, [the Miner], prior to his death was disabled and could not perform his usual coal mining work and this disability was the result of cardiac disease, unrelated to his lungs. It was the result of atherosclerosis.

(WEX 7).

Dr. Ben V. Branscomb, a former B-reader who is board-certified in Internal Medicine, issued a report, dated July 6, 2004, in which he reviewed available medical records (MEX 4, 5). Following a discussion of the Miner's history, numerous clinical tests results, the Miner's exposure to coal mine dust during 35 years of mining, cigarette smoking history, and various medical opinions, Dr. Branscomb opined:

> **CONCLUSIONS:** I concur in the medical opinion that simple CWP is sometimes disabling, that CWP can be a progressive disorder first manifest after mining stops, that its manifestations may be latent, and that sometimes coal mine dust or CWP produce obstructive manifestations. I also incorporate in my definition of CWP for this report the concept that any pulmonary disorder or impairment in any way caused or significantly aggravated by either coal mine dust or CWP is regarded as pneumoconiosis. Further, I accept the concept of disability caused by a non-occupational disorder which has been materially worsened by either coal mine dust or CWP is included as a disability attributable, at least in part, to CWP.
>
> My conclusions which follow are made with a high level of medical certainty or probability.
>
> Please refer also to the summary at the beginning of this communication. A careful examination of the medical records indicate the absence of CWP, medical or legal. Asthma was definitely present and is not severe. It did not progress significantly over the years and responded extremely well to treatment. [The Miner's] pulmonary disease, asthma, or if present CWP played no role in his death. In my judgment the severity of the pulmonary disease under treatment was insufficient to have prevented his last job of one year's duration.

(MEX 4; *see also* WEX 3).

Dr. David M. Rosenberg, a B-reader who is board-certified in Internal Medicine, Pulmonary Disease, and Occupational Medicine (MEX 8), issued a report, dated July 20, 2004 (MEX 6), in which he reviewed and analyzed the then available evidence, and opined:

> In **SUMMARY**, at the time of [the Miner's] death, he was 74 years of age. He had a long smoking history through most of his adult life, and worked 35 years in the coal mine industry. He was treated for hyperlipidemia and hypertension over the years, as well as chronic obstructive pulmonary disease with an asthmatic component. At times he had severe airflow obstruction to a disabling level, but with treatment for his hyperactive component, his pulmonary function tests demonstrated marked improvement over time. His chest x-rays did not reveal micronodularity related to past coal dust exposure. Blood gas studies demonstrated preserved oxygenation for his age, without exercise induced hypoxemia. He was noted to have a positive stress test for ischemia at the time of Dr. Rasmussen's evaluation accompanied by chest pain.

(MEX 6, p. 11). Following further discussion of the evidence, Dr. Rosenberg opined:

> In **CONCLUSION**, it can be stated with a reasonable degree of medical certainty that [the Miner] did not have CWP or associated impairment. While he had significant obstructive lung disease his obstructive impairment improved over time, and clearly was not at a disabling level based on the later pulmonary function studies reviewed in his file. He did have an oxygenation abnormality, and the events surrounding his death were related to arrhythmia which was not caused or hastened by the inhalation of coal mine dust exposure. Obviously, he had coronary artery disease and ischemia, as was noted by Dr. Rasmussen. This condition was one of the general public, and he had risk factors for its development, including hypertension, hyperlipidemia and a family history of heart disease. This underlying heart disease was not caused or hastened by the past inhalation of coal mine dust exposure. Even if [the Miner] was found to have CWP, my opinions with respect to his impairments, disability and death would not change.

(MEX 6, pp. 12-13; *see also* WEX 4). Dr. Rosenberg reiterated the foregoing opinion in his deposition testimony on September 23, 2004 (MEX 12; *see also* WEX 10).

In a supplemental report dated August 8, 2005, Dr. Rosenberg reviewed and analyzed various additional information provided by Employer's representative, including his own deposition transcript and Dr. Perper's report dated June 18, 2005. Following further discussion of the evidence, Dr. Rosenberg opined:

> In **CONCLUSION**, it can be stated with a reasonable degree of medical certainty, that [the Miner], despite what Dr. Perper had outlined, did not have clinical or legal CWP. While he had significant obstructive lung disease, this obstruction was not caused or hastened by past coal dust exposure. Also, he did not have progressive and latent disease, and the events surrounding his death were related to arrhythmia which was not caused or hastened by past coal dust exposure or the presence of CWP. His coronary artery disease caused his arrhythmia and his ultimate demise. As was objectively demonstrated by Dr. Rasmussen on a stress test, [the Miner] had life-threatening coronary artery disease. This is a condition of the general public, and does not relate to past coal dust exposure. [The Miner] would have died in a similar situation, irrespective of his past coal mine dust exposure.

(MEX 13; *see also* WEX 11).

Dr. James R. Castle, a B-reader who is board-certified in Internal medicine and Pulmonary Disease (MDX 8), issued a report dated July 27, 2004 (MEX 7), in which he reviewed and analyzed available evidence. Following a lengthy discussion of the evidence, Dr. Castle stated, in pertinent part:

> …[I]t is my opinion with a reasonable degree of medical certainty that [the Miner] did not suffer from coal workers' pneumoconiosis. He did not have the physical findings, the radiographic findings, the physiological findings, or the arterial blood gas findings to indicate the presence of coal workers' pneumoconiosis.
>
> [The Miner] might have been disabled during life as a result of his bronchial asthma. However, he did not suffer a disabling respiratory impairment due to coal mine dust or coal workers' pneumoconiosis. It is my opinion that prior to his death he was very likely totally disabled as a result of both his age and coronary artery disease. Both of these are conditions of the general public and are unrelated to coal mine dust exposure and coal workers' pneumoconiosis.
>
> [The Miner] was apparently found unconscious and unresponsive at home. There is no evidence to indicate that he had been having any respiratory distress or respiratory symptoms and in fact the recent outpatient notes indicated that he had been doing better than he had in years. CPR was instituted and he was transported to the hospital where he was found to be in asystole. He was subsequently pronounced dead. It is my opinion based after a review of all the information available, that [the Miner] died of either a sudden cardiac arrhythmia related to his underlying cardiac disease or an acute myocardial infarction. There is no evidence to indicate that he had an ongoing acute respiratory problem or hypoxemia. The record clearly does demonstrate that he had evidence consistent with coronary artery disease manifested by chest pain and abnormal electrocardiogram at the time of Dr. Rasmussen's evaluation. He did not demonstrate hypoxemia at rest or with exercise on that occasion. Therefore, for the reasons stated above, it is my opinion that his death was not caused by, contributed to, or hastened by coal mine dust exposure or coal workers' pneumoconiosis. It is my opinion that he died as and when he would regardless of his occupational history.
>
> Even if one were to conclude that [the Miner] did have radiographic evidence of simple coal workers' pneumoconiosis, my opinion concerning his impairment, disability, and cause of death would remain unchanged. My opinion is not predicated upon his normal or negative chest x-ray. It is, however, contingent upon his not demonstrating the physiologic or arterial blood gas findings indicating the presence of coal workers' pneumoconiosis. If coal workers' pneumoconiosis were present radiologically or pathologically, it was in such an insufficient quantity that it did not impact upon his ventilatory function or blood gas transfer mechanisms. For the reasons stated above, it is also my opinion that it could not have caused or hastened his death. His death was sudden and due to a cardiac arrhythmia which might have been brought about by an acute myocardial infarction.

(MEX 7; *see also* WEX 5).

Dr. Gregory J. Fino, a B-reader who is board-certified in Internal Medicine and Pulmonary Disease, issued a supplemental report dated August 26, 2004 (MEX 10). He had previously reviewed the record in a report dated April 18, 1988 (MEX 1). In the report, dated August 26, 2004, Dr. Fino set forth Claimant's work history and background information, the results of various clinical studies, including numerous chest x-ray readings, pulmonary function and arterial blood gases, which were charted, the reported occupational and smoking histories, office records, and findings by various physicians. For the purpose of his analysis of the cause of death, Dr. Fino stated that he would assume that all of the Miner's lung disease was related to coal mine dust. In summary, Dr. Fino opined:

> Taking that into consideration, I cannot attribute his death, in whole or in part, to lung disease. One must understand that when someone "dies in his or her sleep," which was described by Dr. Mitchell as having occurred in this case, death is generally – if not always – due to a heart attack. There was no autopsy in this case. There was a history of hypertension and hyperlipidemia, both of which are risk factors for heart disease. Even though this man had lung disease, without an autopsy one cannot reasonably exclude such diagnoses as a heart attack, or a large stroke, or even a ruptured abdominal aortic aneurysm.
>
> What is interesting to note in the medical literature is the lack of evidence that chronic hypoxemia due to coal mine dust inhalation increases the risk of sudden death,
>
> There is no increased incidence of coronary artery disease as a result of coal workers' pneumoconiosis. There is no increased incidence of coronary artery disease in individuals who have worked in the coal mining industry.

(MEX 10, p. 10; *see also* WEX 8). Following his further discussion of medical literature he concluded:

> …Even assuming that he was totally disabled due to coal dust inhalation, I can state with a reasonable degree of medical certainty that this man's death was not caused, contributed to, or hastened by the inhalation of coal mine dust.

(MEX 10, pp. 14-15). Dr. Fino also addressed the disability causation issue, stating, in pertinent part:

> My opinion regarding the etiology of his disabling lung disease is not that coal mine dust inhalation played a role. The variable obstruction, the improvement with bronchodilators, the lack of a decrease in oxygenation as noted by Dr. Rasmussen in 2000, the office records that noted intermittent wheezing all point to asthma or a smoking-related hyperactive airways disease.

(MEX 10, p. 15). Dr. Fino reiterated the foregoing opinion in his deposition testimony on September 14, 2004 (MEX 11 *see also* WEX 9).

In a supplemental report, dated August 16, 2005, Dr. Fino reviewed additional medical data provided by Employer's representative; namely, Dr. Perper's report, dated June 18, 2005, which included criticism of Dr. Fino's prior report (MEX 14). Dr. Fino responded:

> I am not going to do a sentence by sentence critique of the report by Dr. Perper. I would rely on my report of August 26, 2004, and my testimony from September 14, 2004.
>
> I would note that Dr. Perper stated that this man had pulmonary insufficiency and hypoxemia due to coal mine dust inhalation. He then went on to say that, through hypoxemia, a cardiac arrhythmia occurred. My challenge to Dr. Perper is to show me any evidence of chronic hypoxemia in this case. There was a pO2 of 67 in 1999, but a pO2 of 67 is of no clinical significance. More importantly, 10 months later, in July of 2000, this man had normal resting and exercise arterial blood gases. Therefore, it is pure speculation to state that this man had any hypoxemia.
>
> The bottom line is that it would be purely speculative to be of the opinion that lung disease, regardless of cause, was a significant or material contributing factor in this man's demise. No additional evidence regarding his death is provided.
>
> This additional medical information has not caused me to change any of my opinions.

(MEX 14; *see also* WEX 12).

Conclusions of Law and Discussion

Pneumoconiosis

Section 718.202 provides four means by which pneumoconiosis may be established. Under §718.202(a)(1), a finding of pneumoconiosis may be made on the basis of the x-ray evidence. The clear preponderance of the chest x-ray evidence submitted in conjunction with the current, duplicate Miner's claim is negative for pneumoconiosis. Therefore, Claimant has not established the presence of pneumoconiosis under §718.202(a)(1).

Under §718.202(a)(2), a finding of pneumoconiosis may be made on the basis of biopsy or autopsy evidence. In the absence of any such evidence, this subsection is not applicable.

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found applicable. In the instant case, the presumption of §718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is inapplicable to claims filed after January 1, 1982. The presumption of §718.306 does not apply to claims of a miner who died after 1978. Therefore, the Claimant cannot establish pneumoconiosis under §718.202(a)(3).

Under §718.202(a)(4), a determination of the existence of pneumoconiosis may be made if a physician exercising reasoned medical judgment, notwithstanding a negative x-ray, finds that the miner suffers from pneumoconiosis as defined in §718.201. Pneumoconiosis as defined in §718.201 means a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both "Clinical Pneumoconiosis" and "Legal Pneumoconiosis." §718.202(a)(1) and (2).

The Miner's duplicate claim contains medical records from Clinch Valley Physicians, Inc., including progress notes primarily by Dr. Mitchell dated September 30, 1994 through June 18, 2001, and office notes by Dr. Iosif, a pulmonary specialist, dated September 22, 1999 through September 21, 2000 (WDX 13, 14, 15, 18; CX 2). The Miner's record also contains Dr. Rasmussen's report dated July 10, 2000 (WDX 12), the hospital records on or about the date of the Miner's death (WDX 13; WEX 2), the Miner's death certificate (WDX 8), and, the medical opinions of Drs. Mitchell (WDX 18), Perper (CX 3; MEX 15), Zaldivar (WDX 17), Branscomb (MEX 4, 5), Rosenberg (MEX 6, 8, 12, 13), Castle (MEX 7, 8), and Fino (MEX 10, 11, 14. A qualitative assessment of the conflicting medical opinion evidence must be conducted by analyzing the credibility of each medical opinion considered as a whole, in light of that physician's credentials, supporting documentation and supportive pertinent reasoning.

The office notes by Drs. Mitchell and Iosif list coal workers' pneumoconiosis among numerous diagnosed conditions. However, in both cases the underlying rationale for the diagnosis is neither well-reasoned nor well-documented. The office notes cite the Miner's dramatic improvement in his pulmonary and respiratory condition, which is inconsistent with the progressive and irreversible nature of pneumoconiosis. Therefore, even though Drs. Mitchell and Iosif treated the Miner, their diagnosis of pneumoconiosis, as set forth in the office notes, is accorded little weight. Dr. Rasmussen's recent report is accorded less weight because it is based primarily upon a questionable positive chest x-ray reading, and Dr. Rasmussen's opinion is essentially the same as the one he provided in 1987, which was deemed unpersuasive by Judge O'Brien in light of better reasoned and documented medical opinions by other physicians who found no pneumoconiosis, and was affirmed by the BRB and the U.S. Court of Appeals for the Fourth Circuit (MDX 1).

The Tazewell Community Hospital records reveal that the Miner died suddenly on July 24, 2001, of cardiopulmonary arrest due to a possible, acute myocardial infarction. These records do not list a diagnosis of pneumoconiosis. Although "COPD" is listed under past history, the records do not specify the etiology of the Miner's COPD. Accordingly, such a finding, even if credited, would not establish "legal" pneumoconiosis. The death certificate was signed by Dr. Mitchell, who treated the Miner for several years prior to his death. However, the death certificate is neither a well-reasoned nor well-documented assessment of the cause of death. Moreover, it is equivocal, because Dr. Mitchell listed the immediate cause of death as "*Probable* ventricular arrhythmia" due to COPD (with) coal worker's pneumoconiosis," without explaining the basis for his findings, and without the benefit of an autopsy. [Emphasis added]. Therefore, the death certificate is accorded little weight.

Similarly, Dr. Mitchell's letter, dated August 17, 2002, has little probative value. Dr. Mitchell opined that the Miner "*probably*" died from ventricular arrhythmias, and added that the

Miner's "death was hastened by coal workers' pneumoconiosis since the pulmonary impairment associated with coal workers' pneumoconiosis and lack of oxygen definitely predisposes a person to ventricular arrhythmias. It is *likely* that he would have lived longer had his lungs not been compromised by the Black Lung." (Emphasis added)(WDX 18). However, Dr. Mitchell's "analysis" is rather cursory, equivocal, and unpersuasive.

Consequently, the crux of the Miner's duplicate claim rests on the relative weight accorded the opinions of Drs. Perper (CX 3; MEX 15), Zaldivar (WDX 17), Branscomb (MEX 4, 5), Rosenberg (MEX 6, 8, 12, 13), Castle (MEX 7, 8), and Fino (MEX 10, 11, 14). Facially, all of the opinions appear to be well-reasoned and well-documented. However, the opinions of Drs. Zaldivar, Branscomb, Rosenberg, Castle, and Fino are far more persuasive than the contrary opinion of Dr. Perper. Although Dr. Perper is a well-credentialed forensic pathologist, he lacks the expertise in pulmonary disease of these other physicians. This discrepancy is particularly significant in this case, where pathology evidence is not in issue. Moreover, Dr. Perper relied, in significant part, upon the Miner's work history, a questionable analysis of the x-ray evidence, and the erroneous presumption that the Miner's pulmonary condition had progressively declined. Thus, the medical opinions of Drs. Zaldivar, Branscomb, Rosenberg, Castle, and Fino have greater probative value than that of Dr. Perper, because the other physicians have demonstrably greater expertise in the field of pulmonary medicine, and their opinions are more consistent than Dr. Perper's with the preponderance of the credible, negative x-ray evidence and the pulmonary improvement disclosed by the record and inconsistent with the progressive and irreversible nature of pneumoconiosis. Therefore, Claimant has not established pneumoconiosis under §718.202(a)(4), or by any other means.

Weighing all the relevant evidence together under §718.202(a) to determine whether the Miner suffered from pneumoconiosis, as defined in §718.201, this tribunal finds that the x-ray evidence is negative for pneumoconiosis, and, that the medical opinion evidence does not establish clinical or legal pneumoconiosis. Thus, pneumoconiosis has not been established under §718.202(a). *See, Island Creek Coal Co. v. Compton*, 211 F. 3d 203, 2000 WL 524798 (4th Cir. 2000); *Penn Allegheny Coal Co. v. Williams,* 114 F. 3d 22 (3d Cir. 1997).

Causal Relationship

Since Claimant has not established the presence of clinical or legal pneumoconiosis, she also cannot establish that the disease arose from the Miner's coal mine employment. If Claimant had established the existence of pneumoconiosis, however, she would be entitled to invoke the rebuttable presumption that the disease arose from the Miner's more than ten years of coal mine employment. §718.203. However, on this record, the issue is moot.

Total Disability

The final denial of the prior claim was not based upon "total disability" issue (MDX 2). Therefore, that issue is moot with respect to whether there has been a material change in conditions under §725.309.

Total Disability Due to Pneumoconiosis

Since Claimant has failed to establish the presence of pneumoconiosis, she cannot establish total disability *due to pneumoconiosis* under §718.204(c). With respect to the Miner's duplicate claim, only Dr. Rasmussen specifically attributed the Miner's total pulmonary or respiratory disability, at least in part, to coal mine dust exposure.[8] In so finding, Dr. Rasmussen cited cigarette smoking and coal mine dust exposure as the two primary risk factors, and concluded that "the latter is a contributing factor." However, little weight is accorded Dr. Rasmussen's opinion, because he provided only a cursory "analysis" in reaching his conclusion. Moreover, Dr. Rasmussen's overall opinion is based largely upon a questionable positive x-ray reading, and is further undermined by his own reported findings of "normal" resting blood gases and significant improvement after bronchodilator therapy. Dr. Rasmussen's opinion is also substantially outweighed by the better reasoned and documented medical opinions of the multiple board-certified pulmonary specialists, Drs. Zaldivar, Rosenberg, and Castle, as well as the opinion of Dr. Branscomb, which are more consistent with the credible, negative x-ray evidence and the improvement shown in the Miner's pulmonary condition after bronchodilator therapy.

Conclusion

The relevant evidence in the Miner's duplicate claim does not establish the presence of clinical or legal pneumoconiosis; that the disease arose out of coal mine employment; or that the Miner's total disability was due to pneumoconiosis. Therefore, Claimant has not established a material change in conditions under §725.309, or entitlement to benefits based upon the Miner's claim.

II. Survivor's Claim

Because the Widow's claim was filed on September 10, 2001 (WDX 2), the evidentiary limitations set forth in §725.414 are applicable. Accordingly, with respect to the Widow's claim, the record consists of the hearing transcript, Director's Exhibits 1 through 40 in the Widow's claim file (WDX 1-40), Employer's Exhibits A (EX-A) and 1 through 13 for Widow's claim (WEX 1-13), Claimant's Exhibits A (CX-A) and 1 through 5 (CX 1-5), and Joint Exhibits 1 and 1A (JX 1 & 1A). Director's Exhibits 1 through 24 in the Miner's claim file (MDX 1-24) and Employer's Exhibits 1 through 15 related to the Miner's claim (MEX 1-15) are excluded. However, as previously noted, the medical evidence contained in Director's Exhibits 1 through 24 of the Miner's claim is primarily older evidence which is less probative regarding the existence of pneumoconiosis more than recent evidence, and such evidence does not address the cause of death. Furthermore, almost all of the documents contained in the Miner's case as Employer's Exhibits 1 through 15, are duplicated in Employer's Exhibits 1 through 13 of the Widow's claim.

Except for the medical evidence expressly excluded with respect to the Widow's claim, all of the medical evidence previously discussed in relation to the Miner's duplicate claim, as

[8] Drs. Mitchell and Perper addressed the cause of death.

well as the analysis of the admissible medical evidence, is incorporated in the record of the Survivor's claim.

Even with the exclusion of some negative x-ray evidence, such as the negative readings by Drs. Zaldivar and Navani of the July 10, 2000 x-ray, the preponderance of the x-ray evidence, including the interpretations by B-readers or board-certified radiologists, or both, is negative for pneumoconiosis. The three negative x-ray interpretations by Drs. Meyer, Spitz, and Wiot, who are B-readers and board-certified radiologists, outweigh the two positive x-ray readings by Drs. Patel and Alexander, who are similarly credentialed. At best, the x-ray evidence is deemed inconclusive. In any event, Claimant has clearly failed to establish the presence of pneumoconiosis on the basis of a preponderance of the x-ray evidence.

Even if some of the medical reports such as Dr. Zaldivar's report dated June 26, 2002 were excluded because they exceed the evidentiary limitations of §725.414, the clear preponderance of the medical opinion evidence is negative for pneumoconiosis, for the reasons set forth in the discussion in the Miner's case. Moreover, assuming *arguendo* that the Miner had established the presence of simple pneumoconiosis, Claimant would still have the burden of establishing that the Miner's death was due to pneumoconiosis in order to be eligible for widow's benefits under the Act. Since the Widow's claim was filed after the effective date of the new regulations (WDX 2), proof of death due to pneumoconiosis is governed by §718.205(c), as amended. That regulation states, in pertinent part:

> For the purpose of adjudicating survivor's claims filed on or after January 1, 1982, death will be considered to be due to pneumoconiosis if any of the following criteria is met:
>
> (1) Where competent medical evidence establishes that pneumoconiosis was the cause of the miner's death, or
>
> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
>
> (3) Where the presumption set forth at §718.304 is applicable.
>
> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.
>
> (5) Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death.

§718.205(c).

There is no evidence in this record that pneumoconiosis is the primary cause of the Miner's death. There is no substantial evidence of complicated pneumoconiosis. Accordingly, Claimant has not established "death due to pneumoconiosis" under the provisions of §718.205(c)(1) and §718.205(c)(3). Therefore, the only remaining issue is whether pneumoconiosis substantially contributed to and/or hastened the Miner's death, as provided in §718.205(c)(2), (4), and/or (5).

The medical evidence most relevant to whether "death due to pneumoconiosis" has been proved includes the hospital records on or about the date of the Miner's death (WEX 2), the Miner's death certificate (WDX 8), and, the medical opinions of Drs. Mitchell (WDX 18), Perper (CX 3; MEX 15), Rosenberg (WEX 4, 10, 11), Fino (WEX 8, 9, 12), Zaldivar (WEX 7), Branscomb (WEX 3), and, Castle (WEX 5).

The hospital records establish that the Miner died a sudden death due to "cardiopulmonary arrest" as a result of a "Possible MI, acute, massive." Although "COPD" is listed under past medical history, the etiology of the disease and its relationship to the Miner's death, if any, is not stated. Therefore, the final hospital records do not establish death due to pneumoconiosis.

The Miner's death certificate signed by Dr. Mitchell, and Dr. Mitchell's report dated August 17, 2002, if credited, might support a finding of death due to pneumoconiosis. However, neither the death certificate nor Dr. Mitchell's report is well-reasoned or provides a well-documented opinion. Rather, the death certificate and Dr. Mitchell's report are equivocal, and provide, at best, a cursory analysis of the evidence. Moreover, Dr. Mitchell has not been shown to have credentials as a pulmonary specialist, so that, even though Dr. Mitchell apparently treated the Miner for several years prior to his death, his opinion has little probative value in the contest of this case.

Accordingly, the crux of the Widow's claim rests on the relative weight accorded to the other physicians' opinions; namely, those of Drs. Perper (CX 3), Rosenberg (WEX 4, 10, 11), Fino (WEX 8, 9, 12), Zaldivar (WEX 7), Branscomb (WEX 3), and, Castle (WEX 5). Of those physicians of record, only Dr. Perper related the Miner's death to pneumoconiosis or coal mine dust exposure. For the reasons previously outlined, greater weight is accorded to the opinions of Drs. Rosenberg, Fino, Zaldivar, Branscomb, and Castle, who found that, even assuming the presence of simple pneumoconiosis, the disease and coal mine dust exposure played no role in causing, contributing to, or hastening the Miner's death. That group of physicians has superior pulmonary credentials to those of Dr. Perper, who is a forensic pathologist. Moreover, the opinions of Drs. Rosenberg, Fino, Zaldivar, Branscomb, and Castle are more consistent with the hospital records at the time of a death indicative of a sudden cardiac-related syndrome, as well as the Miner's history of improvement shown after bronchodilator treatment, his history of coronary artery disease, the absence of hypoxemia on recent arterial blood gas studies, and the preponderance of negative chest x-ray evidence. Accordingly, Claimant has also failed to establish that the Miner's death was due to pneumoconiosis under §718.205(c)(2), (4), and (5), or by any other means.

Conclusion

Since the evidence in the Survivor's claim does not establish the presence of pneumoconiosis or that pneumoconiosis caused, substantially contributed to, or hastened the Miner's death, Claimant is not entitled to survivor's benefits under the Act and applicable regulations.

Attorney's Fees

The award of an attorney's fee under the Act is permitted only in the cases in which Claimant is found to be entitled to benefits. Since benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for services rendered to her in pursuit of this claim.

ORDER

The claim on behalf of A. M., the deceased coal Miner, and the claim of M.M., his surviving spouse, for black lung benefits under the Act are denied**.**

A

Edward Terhune Miller
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with this Decision and Order you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which this Decision and Order is filed with the district director's office. *See* 20 C.F.R. §§725.458 and 725.459. The address of the Board is: ***Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, D.C. 20013-7601.*** Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. §802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor for Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Avenue, N.W., Room N-2117, Washington, D.C. 20210. *See* 20 C.F.R. §725.481.

If an appeal is not timely filed with the Board, this Decision and Order will become the final order of the Secretary of Labor pursuant to 20 C.F.R. §725.479(a).